REMARKS

Favorable reconsideration is respectfully requested in view of the foregoing amendments and the following remarks.

Claim 40 has been amended to more particularly define the present invention. Support for the claim amendment is readily apparent from the teachings of the specification and the original claims. Specific support can be found on page 17 of the specification.

With regard to the rejection of claims 40-47 under 35 USC § 112, first paragraph, this rejection is deemed to be untenable and is thus respectfully traversed. Claim 40 has been amended to specify the host to which the apoptosis inducing agent is administered. Further, the teachings on page 15, line 14, to page 17, line 25, of the specification clearly establish the enablement of the claimed method *in vivo*. The noted disclosure of the specification teaches in detail the preparation, administration routes and dose of the apoptosis inducing agent (as a pharmaceutical composition) in the method of the present invention. A person of ordinary skill in the art can clearly practice the claimed method *in vivo* without undue experimentation on the basis of this disclosure. Thus, in view of the teachings of the specification, this rejection cannot be sustained and should be withdrawn.

With regard to the rejection of claims 40-47 under 35 USC § 112, second paragraph, this rejection is deemed to be untenable in view of the amendment to claim 40 and is thus respectfully traversed. As stated above, claim 40 has been amended to specify the subject to which the apoptosis inducing agent is administered. Thus, since the amendment to claim 40 addresses the Examiner's concerns, this rejection can no longer be sustained and should be withdrawn.

With regard to the rejections of the recited claims under 35 USC § 102 as being anticipated by Winget (USP 5,620,962), Yazawa et al. (of record) or Nojima et al. (JP 60-19716) as set forth in items 2-4 of the August 28, 2001 Official Action, these rejections are deemed to be untenable and are thus respectfully traversed.

The Examiner states that these references teach a method for treating inflammation or a method for treating cancers, both of which comprise administering glyceroglycolipid. From the teachings of these references, the Examiner believes that the present invention is anticipated since the prior art methods would **inherently** possess the process of inducing apoptosis. However, Applicants strongly believe that the Examiner is misapplying the principle of inherency under U.S. practice.

Under U.S. case law, a chemical compound or composition and its properties are inseparable. *In re Papesch, 315 F.2d 381, 137 U.S.P.Q. 43, 51 (C.C.P.A. 1963)* Thus, it is well known under U.S. practice that claiming a previously unknown property of a prior art compound or composition will not impart patentability to that compound. *In re Best, 562 F.2d 1252, 1255, 195 U.S.P.Q. 430, 433 (C.C.P.A. 1977)* Likewise, a prior art structure that necessarily functions within the scope of a claimed method anticipates the claim. *In re King, 801 F.2d 1324, 1326, 231 U.S.P.Q. 136, 138 (Fed. Cir. 1986)*

However, the present invention is directed to a new method of use (inducing apoptosis) for a compound (glycerolipid and/or glyceroglycolipid) and not to the compound itself. Thus, since the prior art references, in this case, do not teach a prior art compound or structure, Applicants strongly believe that the principles of inherency is inapplicable in this case.

Under § 2112.02 of the Manual of Patent Examining Procedure (MPEP), in method of use claims, new and unobvious uses of old structures and compositions (or compounds) may be patentable. In other words, the discovery of a new use for a compound based on unknown properties of the compound might be patentable to the discoverer as a method of using. *In re Hack, 114 USPQ 161, 163 (CCPA 1957).*

It is clear from § 2112.02 of the MPEP that the Patent Office has taken a position that a new use for an old product could in fact be claimed as a method. This position is well supported under U.S. statutory and case law. In 35 U.S.C. § 100(b) a "process" is defined as a "process, art or method, and includes a ***new use*** of a known process, machine, manufacture, composition of matter, or material." (Emphasis added.) Further, in *Ex parte Wagner*, 88 U.S.P.Q. 217 (Pat. Off. Bd. App. 1950), the Board of Appeals stated that

> "[M]any processes which are old in a ***procedural sense*** become ***new*** when, by the use of a different (but known) agent, a ***new result*** is accomplished. In considering the patentability of such processes, it appears that ***the real criterion is not whether the steps themselves are shown in the prior art*** but whether the use of the material in the process claimed is suggested by the prior art. It is not considered proper to disregard the specific nature of the material employed in the claimed process which is responsible for the unobvious result and determine patentability of the process solely on the novelty of the physical manipulative steps recited. If the result of the process is unobvious and the particular use of the material is not suggested by the prior art, the process claims should be allowed." (Emphasis and clarification added.)

The prior art methods of Winget, Yazawa et al. and Nojima et al. are based on anti-inflammatory action and anti-cancer action of glyceroglycolipid. It has been known that various physiological actions may be involved in anti-inflammatory action and that various mechanisms may be involved in anti-cancer action. With regard to anti-inflammatory actions, involvement of

blocking of signal transmission via inflammatory mediator, inhibition of biosynthesis of prostaglandin, or suppression of leukocyte invasion to inflammatory location etc. has been suggested. With regard to anti-cancer actions, involvement of alkylation of DNA, inhibition of DNA synthesis, or activation of immune system against cancer etc. has been suggested.

Thus, since the cited references teach a ***completely different use*** (method of treating inflammation and cancers) for glyceroglycolipid ***under a different mechanism***, it cannot be concluded that the presently claimed method of inducing apoptosis (which is a new use and which produces a new result) is inherently taught by the cited references, especially since the apoptosis inducing action (previously unknown characteristic) of glycerolipid and/or glyceroglycolipid is not expressly disclosed or suggested anywhere in the cited references.

Winget discloses an anti-inflammatory action of glyceroglycolipid. However, as stated above, it has been known that various physiological actions may be involved in anti-inflammatory action and none of them, as far as the Applicants know, involve or related to apoptosis. Thus, Winget neither discloses nor suggests the new use (apoptosis inducing) set forth in the presently claimed method.

Likewise, the method of treating cancers taught by Yazawa et al., is based on the ***inhibitory action of glyceroglycolipid on carcinogenic promoters***. Such action is not at all related to the apoptosis inducing action of the present invention.

Lastly, Nojima et al. teach an antitumor agent containing glyceroglycolipid. As to the mechanism of action thereof, Nojima et al. disclose that it may be due to host-mediated activation of the immune system. In addition, Nojima et al. describe that the glyceroglycolipid used in their

study ***did not exhibit any cytoxicity***. Thus, Nojima et al. clearly teach away from the apoptosis inducing action of glyceroglycolipid of the present invention.

Further, in order to judge whether its compound has antitumor action, Nojima et al. studied whether the compound exhibits life-elongation effect on patients suffering from spontaneous cancer, carcinogen-induced cancer, or grafted cancer, etc. However, Nojima et al. does not disclose or suggest at all that apoptosis is the mechanism involved in such life elongation effect. Since, as stated earlier, various mechanisms may be involved in anti-cancer action, Nojima et al. clearly do not teach or suggest the new method of use (apoptosis inducing) set forth by the present application.

Thus, for the reasons set forth above, these rejections under 35 USC § 102 cannot be sustained and should be withdrawn.

With regard to the rejection of claims 40-47 under 35 USC § 103(a) as being unpatentable over both Winget (USP 5,620,962) and Yazawa et al. (IDS, AB), Nojima et al. (JP 60-19716, IDS, AA) in view of Nakai at al. (USP 5,672,603) and Nelson ("Isolation and Purification of lipids from Biological Matrices," in Analysis of Fats, Oil and lipoproteins, Edited by Edward G. Perkins, 1993), this rejection is deemed to be untenable and is thus respectfully traversed.

To establish a *prima facie* case of obviousness, the Patent Office must satisfy three requirements. First, the prior art relied upon, coupled with the knowledge generally available in the art at the time of the invention, must contain some suggestion or incentive that would have motivated the skilled artisan to modify a reference or to combine references. Secondly, the proposed modification of the prior art must have had a reasonable expectation of success,

determined from the vintage point of the skilled artisan at the time the invention was made. Lastly, the prior art references or combination of references must teach or suggest all the limitation of the claims.

Here, in this case, there is no suggestion or incentive that would have motivated the skilled artisan to modify the cited references or to combine the cited references. As it is well known under U.S. practice, motivation is lacking when the state of the art at the time of the invention pointed researchers in a different direction than the inventor proceeded.

The Examiner has reasoned that since Nakai et al. teach that apoptosis is involved in cancer treatment when the cancer cells are killed and that apoptosis regulating compounds or composition are useful as anticancer agents, it would have been obvious to one skill in the art to use anticancer agents containing glyceroglycolipid (as taught in, for example, Yazawa et al.) for inducing apoptosis. Thus, the Examiner has based his obviousness rejection on the relationship that glyceroglycolipid has an anticancer effect (Yazawa et al. and Nojima et al.) and that apoptosis is a mechanism for killing cancer.

However, both of the cited references, Yazawa et al. and Nojima et al., teach away from using glyceroglycolipid in a method for inducing apoptosis since, as stated earlier, both references teach a mechanism ("inhibitory action of glyceroglycolipid on carcinogenic promoters", "host-mediated activation of the immune system" "no cytoxicity") unrelated to the apoptosis inducing action of glyceroglycolipid. It should be noted that by teaching away from the present invention, the cited references, Yazawa et al. and Nojima et al., sever the logical relationship which form the basis of this obviousness rejection. Further, in view of the fact that various mechanisms may be

involved in anti-cancer action, the fact that glyceroglycolipid has an anticancer effect cannot motivate one skilled in the art to use glyceroglycolipid in a method for inducing apoptosis especially since Yazawa et al. and Nojima et al. teach a different mechanism of glyceroglycolipid in treating cancer.

The same argument also holds true for the cited reference of Winget. As stated earlier, Winget discloses an anti-inflammatory action of glyceroglycolipid which is totally unrelated to apoptosis. Thus, one skilled in the art could not possibly have been motivated to combine the teachings of Winget and Nakai et al. to arrive at the present invention since neither reference discloses nor suggests the new use (apoptosis inducing) of glyceroglycolipid.

Further, the proposed combination of the cited references did not have a reasonable expectation of success since from the vintage point of the skilled artisan, it could not have been predicted from the teachings of the cited references that glyceroglycolipid can be use in a new method of inducing apoptosis since such a property is not disclosed in any of the cited references.

Even if it has been known that apoptosis inducing agents are useful as anticancer agents, it cannot be assumed that anticancer agents are useful at inducing apoptosis. As stated above, various mechanisms may be involved in anti-cancer and anti-inflammatory action. Thus, a person of ordinary skill in the art can not predict and thus reasonably expect that an anticancer or antiinflammatory agent such as glyceroglycolipid is also useful in a new method of inducing apoptosis.

It should be noted that the remaining reference, Nelson, merely teaches the use of acid treatment in lipid separation and purification, and therefore, does not cure or address the deficiencies in the teachings of the cited references and the arguments noted above.

Thus, in view of the reasons outlined above, it is clear that claims 40-47 are unobvious over the combined teachings of Winget, Yazawa et al., Nojima et al., Nakai et al., and Nelson and thus, should be withdrawn

Attached hereto is a marked-up version of the changes made to the claims by the current amendment. The attached page is captioned "**Version with markings to show changes made.**"

In view of the foregoing amendments and remarks, it is respectfully submitted that the Application is now in condition for allowance. Such action is thus respectfully solicited.

If, however, the Examiner has any suggestions for expediting allowance of the application or believes that direct communication with Applicants' attorney will advance the prosecution of this case, the Examiner is invited to contact the undersigned at the telephone number below.

Respectfully submitted,

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VERSION WITH MARKINGS TO SHOW CHANGES MADE

Claim 40 has been amended as follows:

40. (Twice Amended) A method of inducing apoptosis comprising administrating an apoptosis inducing agent which comprises glycerolipid and/or glyceroglycolipid as the effective component(s), to an individual.